the approximately 1% complication rate of performing arteriography on all patients with proximity injuries without hard signs. This study alone resulted in a cost of more than $100,000 for performing these examinations on a routine basis, the vast majority of which (95/118) were not needed.

At the University of Florida Health Science Center in Jacksonville, we have been following a prospective protocol outlining the observation of proximity penetrating injuries with no hard signs of vascular trauma for more than 5 years. As time goes on and follow-up continues, the data overwhelmingly convince us that this is the ideal treatment of these patients. Other centers have also prospectively confirmed our results, including Francis et al.,[4] and Weaver et al.[5]

We believe that the article by Tufaro et al. is potentially misleading for the reasons cited above. As with any retrospective study, no conclusions regarding management should be inferred from their data. They must now offer *prospective* data to confirm their retrospective observations and support their conclusions, which are clearly at odds with all other prospective studies of this issue to date. We encourage all centers involved with significant vascular trauma to prospectively delineate the appropriate management of occult arterial injuries, because we remain convinced of the overall benign nature of these lesions, and of the clear benefit of observation to these patients.

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24/41/64192

Regarding "Atherosclerotic aneurysm formation in an in situ saphenous vein graft"

To the Editors:

The authors of a recent case report of aneurysmal degeneration of a saphenous vein graft may have missed the interesting point of their own observations (Alexander JJ and Liu Y-C; J VASC SURG 1994;20:660-4). They argue that the case supports the view that atherosclerosis causes aneurysms, because the pathologic condition of the vein graft showed atherosclerotic changes. Almost all aneurysms show atherosclerotic changes, even those associated with cervical ribs, Marfan's syndrome, and coarctations, because the conditions at the flow surface are those that promote atherosclerosis. Of the several cases of aneurysmal degeneration of a vein graft that have come to my attention, all of the grafts had been done to repair aneurysms of the popliteal artery. I believe that the real significance of the observations of the authors is that the susceptibility of patients to form aneurysms is a systemic phenomenon that may affect veins as well as arteries.

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24/41/64194

Combined internal carotid and hypoglossal artery endarterectomies in a symptom-free patient with contralateral internal carotid artery occlusion

To the Editors:

Recently, Fantini et al. reported two patients with symptoms of internal carotid artery stenosis and persistent hypoglossal artery who underwent successful carotid thromboendarterectomy (J VASC SURG 1994;20:995-9). We want to report our experience with an unusual case of a symptom-free patient with a combined internal carotid and hypoglossal artery stenosis associated with an occlusion of the contralateral internal carotid artery. A successful combined internal carotid and hypoglossal endarterectomy was carried on. Justification of the procedure and technical strategies are discussed.

CASE REPORT

A 74-year-old woman with a history of bilateral asymptomatic carotid artery bruit was referred for carotid artery investigation. On duplex and continuous Doppler assessment, the left internal carotid artery was occluded, and the left external carotid artery was severely stenotic (>80% surface area reduction). Both right internal and external carotid arteries presented high-grade stenosis. The left vertebral was patent, but no right vertebral artery was detected. The carotid angiogram confirmed these findings but further disclosed an extracranial internal carotid artery branch taking off 4 cm above the right carotid bifurcation with a 60% (cross-diameter) stenosis identified as a persistent hypoglossal artery (Fig. 1, *A*). The right vertebral and posterior communicating arteries (Fig. 1, *B*) were aplastic, whereas the left vertebral artery was hypoplastic.